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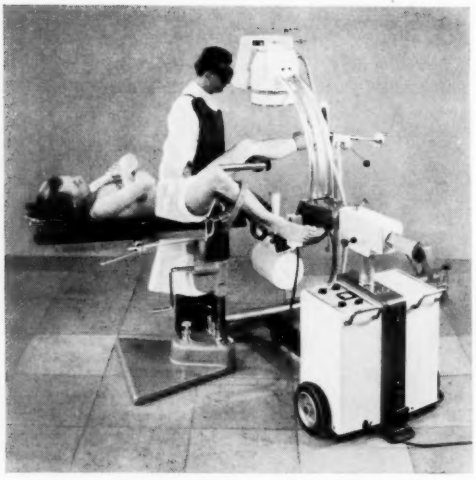

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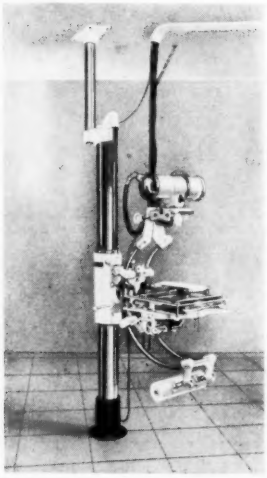

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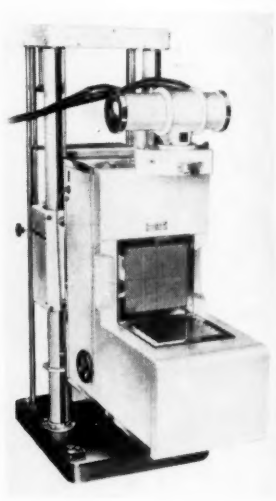

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THE JOURNAL OF THE
CANADIAN ASSOCIATION OF RADIOLOGISTS

Volume VIII December 1957 Number 4

THE MEANING OF SPONTANEOUS REGRESSION*

PART II

WILLIAM BOYD, M.D.
Toronto, Ontario

Reference has already been made to the meaning of the expression "spontaneous". A moment's thought is sufficient to convince us that in biology, as in other fields of science, nothing is really spontaneous, for every event must have a cause. In this instance the word is used in relation to the absence of adequate therapy. The connotation of "regression" demands comment. It must not be confused with remission. The latter term signifies a temporary amelioration of a condition, to be followed later by an exacerbation. Regression, on the other hand, may (or may not) be permanent. It at least suggests possible permanence, i.e., cure.

We are perhaps too demanding in the matter of cure in cases of cancer. If a cancer patient remains free from recurrence for 20 years and is then killed in an accident or dies of an intercurrent infection it is surely straining a point too far to object that if he had lived for one more year the cancer might have recurred. Emphysema and chronic nephritis are truly incurable. with no hope of spontaneous regression.

Cancer cells are not immortal. If they do not divide again after the mitotic division which gives them birth they will die sooner or later (Cowdry[17]). "Limitless growth is not a power possessed by all malignant tumours. The universal employment of surgical measures renders this fact less obvious than it might be" (Ewing[18]). It is of interest to recall that in 1909 Sampson Handley[19] in an address entitled "The Natural Cure of Cancer" enunciated what he called the unrecognized law of cancerous growth: "Every aggregation of carcinoma cells has a definite life cycle, and, after increasing in size for a varying period and at a varying rate, tends spontaneously to undergo degenerative and fibrotic changes." We do not necessarily need to subscribe to Handley's ideas of how these changes are brought about.

There is good reason to believe that the body exercises some control over the growth of cancer cells, just as the growth of all normal cells is also controlled. The difficulty is to suggest the nature of these controls. It is, of course, equally difficult to explain the control of normal growth. To make the difficulty complete, we have no satisfying definition of a cancer cell nor of the cancer process. To every attribute which has been suggested there prove to be exceptions. This is true both in the morphological and the physiological fields. Worthy of consideration in this respect are the subjects of carcinoma in situ, dormant, latent or occult carcinoma, the effect which removal of the primary tumour occasionally has on the metastases, and the hormonal control of malignant growth.

It is well for us to bear in mind that malignancy is essentially a clinical concept, which embodies certain common associations of clinical and pathological characteristics, but which lacks the exactness of a scientific entity. We speak of invasiveness, ability to metastasize, and autonomous growth, yet a highly invasive tumour such as a glioblastoma may never become disseminated, a strictly localized malignant adenoma of the thyroid may manifest its presence by bone metastases, and an apparently autonomous carcinoma of the breast or prostate may respond readily to hormonal control. Each cancer seems to run its own course, and this course may bear no relation to the size of the tumour, the duration of the disease, or the type of treatment given. From these considerations it becomes evident how difficult if not hopeless is the task of attempting to determine the factors which may lead to spontaneous regression.

Carcinoma in situ is that condition in which the cytological appearance of the lesion is indistinguishable from that of classical carcinoma, but without the invasive and metastasizing characteristics associated with the concept of malignancy. Every cancer must first be a carcinoma in situ, but the condition is most readily recognized in the cervix, skin, bronchus, stomach and bowel. There is every reason to think that the lesion may often be the forerunner of classical cancer, but there

*Presented at Annual Meeting of the Canadian Association of Radiologists, Vancouver, B.C., January 17, 1956.

(Part I of this paper appeared in the September issue of the J.C.A.R.)

is also good reason to believe, at least in the case of the cervix, that it may regress and disappear. In this instance hormonal influence is very evident. May this not also be true of many "unsuccessful" cancers? It is only the "successful" ones which become clinically apparent.

Latent or occult cancers have been described in the prostate, the breast, and in other organs. These are picked up on microscopic examination, and the more thorough the examination, the more frequently are they found. Latent cancers of the prostate have been encountered in 25 per cent of men over 50 years of age, and the number increases with each decade. Franks (Imperial Cancer Research Fund) performed autopsies on 220 men over 50 years of age who had died from various causes. In 36 per cent, histological evidence of cancer of the prostate was found. In the 6th and 7th decades a tumour was found in about a third of all cases, and in the 8th decade a tumour was present in nearly half. These figures are, of course, greatly in excess of the incidence of lethal cancers, so it is reasonable to assume that these "malignant" foci fail to develop into clinical cancer. Some influence must hold them in check. Whether or not they undergo regression it is of course impossible to say.

Regression of metastases after removal of the primary tumour is an undoubted fact. Many instances of this phenomenon have been given in the earlier part of this paper. It would appear that the primary tumour exerts some influence on the health of the cells of the metastases, and that when this influence is withdrawn regression may follow in the exceptional case.

Hormonal control of cancer has been extensively studied both in the experimental animal and in man. A masterly review of the subject as it applies mainly to the experimental animal is contained in a recent paper by Noble.[20] These tumours grow with all the characteristics of malignancy, except that their continued growth is dependent on a constant hormonal stimulation. Dependent malignant thyroid tumours may be induced in mice by a variety of procedures, such as thyrotrophic hormone, or anti-thyroid drug or iodine deficiency which induce an increased production of the pituitary thyrotrophic hormone. These tumours cannot be transplanted into normal animals unconditioned by hormonal imbalance; such tumours regress and disappear. In the prostate tumours can be induced either by androgen therapy or by endogenous androgens produced by the interstitial cells of the testis stimulated by pituitary gonadotrophin. In man there is no proof that the initial tumour is the result of increased production of male hormone, but continued growth is certainly controlled by androgen stimulation. In the breast the problem is more complex, for this organ is stimulated both by estrogens and androgens. Some breast tumours seem to be independent of hormonal control, but in these cases the adrenal cortex may be responsible, for while that structure normally produces androgens, it may also secrete estrogens. Noble points out that androgen therapy in breast cancer may have undesirable side-effects, "As the husband remarked on meeting his androgen-treated wife returning from hospital — he didn't know whether to kiss her or to shake hands".

Endocrine tumours, however, may change slowly from the dependent to the independent type of cancer. Noble remarks that the final change from a cell which shows progressive abnormal growth and is yet hormonally dependent to one entirely autonomous is probably the most fundamental problem in cancer research. This change may be due to mutation or more probably to a progressive metabolic adaptation in the cell which has become habituated to an altered hormonal or chemical environment.

Emerson and his associates[21] have studied the morphological changes produced in carcinoma of the human breast by the administration of steroid hormones. They found that the fundamental change in regression was softening, due to a loosening of the dense collagen surrounding the cancer cells, the tangled fibrous strands becoming separated by wide spaces with partial solution of the collagen. The tumour cells were swollen, their cytoplasm vacuolated, their nuclei pyknotic, till finally they disintegrated and disappeared. A few cancer cells could be observed in the lymphatics and ducts, where they were not readily accessible to hormonal influence. A marked infiltration with plasma cells was a constant feature. It is possible that the hormones may enhance a natural defense reaction which is not sufficient of itself. The histological changes occasionally observed in cases of spontaneous regression of cancer are similar to those described above.

Physiological mechanisms

We have seen that hormonal stimulation may incite the growth of certain cancers and that withdrawal of such stimulation may lead to regression. Other physiological mechanisms may either destroy malignant cells or hold them in check for long periods. One such mechanism has been demonstrated experimentally by Horava.[22] A pouch was made in

the subcutaneous tissue of the back of rats by the injection of air. Into this pouch was introduced a suspension of Walker 256 tumour. The tumour grew readily, and growth was accompanied by an accumulation of haemorrhagic odourless fluid, associated with necrosis of the surface of the tumour. In several animals the tumour became stationary at the time the fluid collected (end of second week), but there was a sudden increase in growth when the fluid was withdrawn. Haemorrhagic necrosis of the tumour occurred on injection of the fluid into it. In such a case cancer regression could be due either to substances such as growth-inhibiting steroids originating in the tumour tissue or to response on the part of the host in the form of local inflammation or defensive enzymes.

The chemical make-up of cancer cells may determine the question of regression. Caspersson and Santesson[23] studied the nucleoproteins of tumour cells by the use of ultraviolet microscopy, and concluded that there were two principal types of cells: "A" cells, which are active and aggressive with nuclei rich in desoxyribonucleic acid, and "B" cells, a regressive type with nuclei poor in DNA, but nucleoli rich in ribonucleic acid. There is regression of DNA and accentuation of RNA before the cell dies. Under radiation treatment of radiosensitive cases of carcinoma of the cervix there is a marked drop in A cells and a rise in B cells (Gusberg et al).[24] In this connection the suggestion of Huth[25] is of interest that the remissions of cancers both in mice and men which may be associated with suppuration and the destruction of large numbers of germs may be related to the liberation of ribonucleic acid from the bacteria.

Immune Reactions

The work and ideas of Green on immunological reactions in cancer are of interest in relation to the question of spontaneous regression. It has long been known that the carcinogens of coal tar are weakly tumour-inhibitory for transplanted cancer in the rat. Green[26] has shown that certain non-carcinogenic fractions, polycyclic hydrocarbons isolated from pitch-coking oil, possess this power to much greater degree. Of these the most used is 3-4-benzotetraphene. It must be noted that they have no inhibitory effect on either spontaneous or chemically induced tumours. When the treatment is given during the first three days the transplant continues to grow for a week or more; then regression, usually absolute, sets in. The treatment is powerful in some animals, but powerless in others. Green believes that the tumour-inhibitory compounds modify the antigenic structure of a specific protein complex in the tumour cells, so that it becomes "foreign" to the organism. This isoantigen appears to act by some specific stimulant action on antibody-forming cells of the reticulo-endothelial system, an idea strengthened by the discovery that cortisone completely suppresses this homologous immunity.

The histological changes in the tumour are similar to those seen in spontaneous regression. The tumour-inhibitory substance is not found in the tumour, but only at the site of injection and in the local lymph nodes. There is a great increase in the number of plasma cells in these nodes, and they are believed to be the source of the antibody.

Every pathologist is familiar with the round-cell infiltration of lymphocytes and plasma cells which is often seen immediately subjacent to a carcinoma of the skin or cervix. This infiltration, which is sometimes remarkably intense, is not a defense against infection, for it is most characteristically present before ulceration occurs. The observer gets the impression that some substance is passing from the cancer into the surrounding tissue and there exciting a reaction. Such a histological picture tends to support the concept of an immunological reaction in some cases of cancer.

It seems probable that the wide fluctuations often observed in the rate of growth of any one tumour are due to changes in host resistance rather than to fluctuations in the virulence of the tumour. If the body is capable of some form of defense reaction of an immunological character, the presence of specific antibodies might be expected. It is true that no qualitative chemical difference has been demonstrated between malignant and normal tissue, but this is also true of the group-specific substances of red blood cells, which yet are easily distinguishable immunologically. Nucleoprotein extracts of transplanted tumours appear to be immunologically distinct from those of normal tissues (Maculla).[27] Using complement fixation technique, Graham and Graham[28] have applied this idea to cases of cancer of the female reproductive system. They prepared an antigenic extract, consisting probably mainly of desoxynucleoprotein, from the tumour and were able to demonstrate specific antibodies circulating in the patient's blood in 12 out of 48 cases. These were absent in all the far-advanced cases.

Some of the observed facts in cases of regression of human cancer would seem to fit the concept of an immunological relationship

between tumour and host, activated possibly by some change in the protein complexes of the malignant cells. In many of the reported cases there has been a history of incomplete surgical removal, inadequate radiation, some acute febrile disturbance, or some metabolic change such as artificial or natural menopause. Stewart's case in which there was a sudden and dramatic change in the tumour accompanied by an allergic-like reaction suggests a similar mechanism. Rohdenburg[16] mentions the case of a man, 45 years of age, with widespread lesions of lymphosarcoma. One year after the onset of the illness all the tumours, including the splenomegaly, disappeared overnight. The recession lasted only one week, the tumours returned and reached their original size in three weeks, when the patient died. The time-honoured explanation of inadequate blood supply could not hold water in this case. The fact that periods of spontaneous arrest or regression may alternate with periods of growth point to a natural resistance of the tissues to cancer, just as do the long latent periods during which metastases may lie quiet and the latency of some dormant cancers which has already been discussed. Bowen's disease of the skin has been known to remain static for as long as 40 years, yet at any time it may spread on the surface, invade the deeper tissues, and set up metastases like any other epidermoid carcinoma. All tumours do not possess unlimited growth energy against which the body is incapable of protecting itself. Even though a cancer is widely distributed throughout the body, the patient may continue to live for long periods in good health (Morton and Morton).[29] A delicate balance is established between tumour and host in which neither seems to harm the other.

The whole story of experimental carcinogenesis illustrates the fact that life is not always a bed of roses for the cancer cell. Some tumour transplants flourish, but others regress and die. Induced tumours also pursue a variable course, some regressing and dying. Even in the case of spontaneous animal tumours a fraction of 1 per cent regress and disappear.

Conclusion

Throughout this paper emphasis has been laid on the concept that cancer is not necessarily a completely one-sided process, autonomous in the sense that it is anarchistic, above and beyond all forms of control. When we consider that everyone is exposed repeatedly to numerous cancer-inducing agents and conditions, that indeed we may be said throughout life to swim through a sea of carcinogens, it seems reasonable to suppose that everyone who lives for a number of years must develop one or more foci of neoplasia, just as he develops foci of tuberculous infection. But in both cases these are held in check. In later life they may be detected as latent carcinoma and carcinoma in situ or more rarely as quiescent metastatic deposits.

The desirability of dwelling on such thoughts is that they encourage a different outlook on the cancer problem. In 1910 Sir Alfred Pearce Gould[11] pointed out that the oft-quoted statement that 1 out of every 8 women who reach the age of 35 years will die of cancer may be rephrased by saying that 7 out of 8 women who reach this age will *not* die of cancer. The emphasis is on immunity and resistance rather than on susceptibility. When the clinician and the experimentalist concentrate on immunity rather than on mortality statistics and methods of carcinogenesis, we may begin to think of the prevention, the control and the cure of cancer. In "The Pilgrim's Progress" we read of "a room where was a man with a muck-rake in his hand. There stood also one over his head with a celestial crown in his hand, and proffered him that crown for his muck-rake; but the man did neither look up nor regard, but raked to himself the stones, the small sticks, and the dust of the floor". The body is not so defenseless as the "muckrate" students would have us believe, and perhaps it is time that we stopped using such expressions as "astounding", "incredible", and "unbelievable" when we hear of a case of spontaneous regression of cancer, and concentrate a little on the angel's crown, which is Immunity.

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BOOKS RECEIVED

Books received are acknowledged in this department and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Graphs and Tables for Radiotherapy, by Dr. Felix Wachsmann and Dr. Alexander Dimotsis, Publisher, S. Hirzel, Germany.

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In Memoriam

It is with deep regret that the Canadian Association of Radiologists records the deaths of three radiologists during 1957.

BLACKETT, Arthur Edwin
November 5, 1892, Glace Bay, Nova Scotia
M.D. — Dalhousie University, 1915

COLE, John McKay
July 26, 1901, London, Ontario
M.D. — University of Western Ontario, 1928

NICHOLL, John Frederick
November 11, 1917, Belfast, Ireland
M.D. — Queen's University, Belfast, 1942

A STUDY OF INTRINSIC CARCINOMA OF THE LARYNX TREATED BY COBALT^{60} BEAM THERAPY*

IVAN H. SMITH, M.D., and PAUL M. PFALZNER, M.Sc.**
London, Ontario

This is a study in technique and dosage of intrinsic carcinoma of larynx treated by Cobalt^{60} beam therapy. The time period is too short and the case volume is too small to deduce from a statistical viewpoint, but we believe, considering the rapidly expanding usage of Cobalt^{60} beam therapy and supervoltage that the presentation is timely.

TABLE No. 1

INTRINSIC CARCINOMA OF LARYNX TREATED BY COBALT^{60} BEAM THERAPY

(Observation Period 13 Months to 4½ Years)

Year	Total Cases		Alive	Deceased	Untraced
1952	6	—	5	1	0
1953	5	—	4	0	1
1954	2	—	2	0	0
TOTAL	13	—	11	1	1

The thirteen cases studied represent the entire group of stage 2, and one stage 3, intrinsic laryngeal carcinoma treated during the years 1952, 1953 and 1954 at the Ontario Cancer Foundation London Clinic. In our opinion none was suitable for laryngofissure, but all were technically operable by laryngectomy. Age and general condition precluded surgery in at least two and perhaps three. The age range was forty-four to eighty years with an average of sixty. All were males. All cases were histologically termed "squamous" or "epidermoid" carcinoma. Five were graded as I, five were graded as II, while tissue did not permit grading of three.

Of the thirteen cases, one was staged as 3 with involvement of both true cords, the right false cord, the anterior commissure and with fixation of the arytenoids. Twelve cases were staged as 2, either with anterior commissure extension, fixation to true cord or involvement of false cord; actually this group included six cases with subglottic extension, five of which were detected by tomograms only. There being no internationally accepted staging for carcinoma of the larynx, we suspect that all of our subglottic cases would, in some centres, be categorized as stage 3. This being primarily a study in dosage and clinical response, cases with metastases, or so advanced as to be suitable only for palliation are excluded. This late group is in an entirely different category and should be appraised separately. No stage 1 cases confined to the tissue of origin, such as the true or false cords alone, were registered in the Clinic during this study.

The observation period covers thirteen months to four and one-half years. Eleven of the thirteen are without disease. One is dead. One was free of disease at the end of the first year but cannot be traced.

Table No. 2 portrays a synopsis, case by case, of basic treatment factors and comments on special clinical features.

Influence of Early Observations on Current Therapy

As a result of clinical observations made on this study group we at present aim to deliver 1500 r tumour dose per week to a planned total dose of 6000 r in a four-week period. The reactions are assessed daily during the fourth and fifth weeks when the dosage rate is occasionally decreased and treatment terminated when an apparent "optimum" has been achieved between the 6000 and 7000 r figure.

For the early stage 1 case considerable thought should be given to the use of a single field. We have been tardy in our clinical studies with wedge filters and rotation therapy since our case volume is limited, but the physical advantages of both induce attractive speculations.

Physical Aspects of Treatment Technique

All the patients in this group were treated with the original Eldorado Model A Kilocurie Cobalt^{60} Unit, manufactured by Atomic Energy of Canada Limited.

*Presented at the Eighth International Congress of Radiology, Mexico City, July, 1956.

**From The Ontario Cancer Foundation, London Clinic, London, Canada.

TABLE No. 2

INTRINSIC CARCINOMA OF LARYNX

Résumé by Year and Case of Treatment Factors and Results

Patient	Age	Tumour Dose	Overall Time	No. of Fields	Size of Fields	Comments
1952:						
J.P.	57	4500 r	3 weeks	3	5x6 cm. Ant. 6x6 cm. P.L.	Recurrence 5 months later Retreated single field 6x8 cm. T. D. 5000 r, 3 weeks Well (4 years after initial treatment)
D.S.	55	5000 r	4 weeks	3	10x17 cm.	Cartilage necrosis in 7 months Death in 16 months
J.K.	47	5500 r	3½ weeks	3	8x10 cm. Ant. 6x6 cm. P.L.	Well (3 years 7 months)
G.R.	44	5500 r	3½ weeks	3	10x8 cm.	Well (3 years 9 months)
J.H.	58	5500 r	5 weeks	3	7x6 cm.	Well (4 years 6 months)
P.S.	72	6500 r	4 weeks	3	5x5 cm.	Well (3 years 7 months)
1953:						
L.T.	68	6000 r	5 weeks	3	6x8 cm.	Well (2 years 7 months)
E.B.	42	6000 r	4½ weeks	3	6x7 cm. Ant. 5x7 cm. P.L.	Previous X-ray therapy to larynx Cartilage necrosis Well with fistula (2 years 9 months)
E.Z.	50	6800 r	4½ weeks	3	5x5 cm.	Well (3 years)
J.S.	71	7000 r	4 weeks	1	7x7 cm.	Reported well at 13 months Untraced
P.C.	76	7000 r	4½ weeks	3	5x9 cm.	Well (2 years 1 month)
1954:						
D.V.	63	6100 r	4 weeks	3	5x5 cm.	Well (1 year 1 month)
J.C.	80	6300 r	5½ weeks	3	5x5 cm.	Well (1 year 6 months)

The usual small-field beam directed techniques were employed in the planning and carrying out of the treatment. A plaster of paris collar was made for each patient and localization radiographs were taken with markers affixed to the cast. A plane section through the tumour centre was then chosen and with the aid of the plaster cast the outline of the neck in this plane was drawn on paper. The size and location of the tumour were obtained from measurements made on the radiographs allowing for the magnification factor involved. A three-field technique with one anterior and two posterolateral fields was used in all except one instance where a single oblique field was applied. A complete isodose distribution in the central treatment plane resulting from three such fields is shown for a representative outline in Figure 1. It is to be noted that the posterolateral field entrance portals were built up with wax bolus. The entrance and exit points of each field were marked on the plaster cast which was worn by the patient during treatment. A back-pointer and front entrance cone assured proper beam alignment (Figure 2). Treatments were given to each field for five days per week.

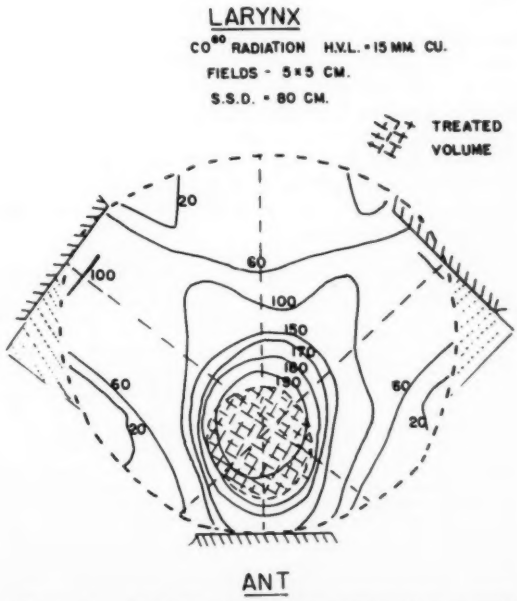

Figure 1. Isodose distribution obtained in a typical three portal larynx treatment with Cobalt^{60}.

Figure 2. Carcinoma of larynx patient in treatment position. Plaster collar, back pointer and perspex cone attached to Cobalt^{60} teletherapy unit. Three-field beam directed technique.

The three-field beam directed technique is convenient, accurate, has served us well, and results in a favourable dose distribution with a tumour dose of as much as 200% of the entrance doses. It should be pointed out, however, that the dose received at the spinal cord ranges from 40% to as high as 60% of the tumour dose depending on the cross-section of the neck region. Should this prove to be too high, two anterior oblique wedge fields could be substituted with advantage.

Reactions

It is our impression that with the dose rate used, fibrinous reactions appeared earlier and regressed more rapidly than when treated by X-ray. It is to be remembered, however, that the dose rate was higher and the overall time shorter than with our X-ray therapy group. Only occasionally did a small patch of moist dermatitis occur anteriorly. Dysphagia to a mild degree was seen, occasionally severe but short-lived.

An excellent average example of reaction and response is as follows:

Case D. V. #54 1165, age 63. This patient had intermittent hoarseness for two years which had persisted during the last six months. Malignant disease involved the right true cord, crossing the commissure to encroach minimally on the left cord. The pathology report was "squamous cell carcinoma, grade II". Using a three-field technique with 5x5 cm. portals, 6100 r tumour dose was delivered in four weeks. Treatments were commenced on September 10, 1954. The following subsequent notes were made: September 28, "there is beginning erythema of the skin anteriorly and early fibrinous reactions over the arytenoids and the epiglottis". October 1, "a fibrinous reaction is appearing on the false and true cords. The lesion is shrinking but not too much soreness so far". October 5, "mild fibrinous reaction is noted in the left larynx proper. There is oedema of the arytenoids". October 7, "treatment completed today. There is light but even fibrinous reaction over the larynx, arytenoids, pharynx and up to the uvula. There is not too much difficulty in swallowing. The tumour has disappeared". October 28, "the patient returns today looking very well. He says that his throat was sore for about two weeks following treatment but there is no soreness now. He is able to eat without reservation and hoarseness disappeared about one week ago. The larynx is easy to see. The right cord is red and perhaps a little swollen. No fibrinous reaction is present. The tumour has completely disappeared. The left cord is perfectly smooth as is the commissure". On January 13, and November 18, 1955, the patient was recorded as without evidence of disease at that time. The cords were slightly injected but perfectly free of disease. The anterior commissure was easily visualized and without pathology. There was no laryngeal oedema. There were no nodes.

Observations on Cartilage Necrosis, 2 Cases:

Case E. B. #53 1104, age 42. As a young man this patient was treated by X-ray therapy for papilloma of the larynx. Otolaryngological examination revealed both cords to be involved with disease extending around the anterior commissure. Both arytenoids were fixed. The right false cord was also involved by disease. This case was staged as 3, the only one in the series so staged and yet the disease remained intrinsic. A three-field technique was used, the central anterior portal was 6x7 cm. the right and left posterolateral portals were 5x7 cm. A dose of 6000 roentgens was delivered in a four-week period to the tumour. Recalculation of dosage indicated the maximum to be 6300 r. Treatment was commenced on August 25, 1953. He developed a heavy fibrinous reaction over the entire larynx involving arytenoids and base of epiglottis. A definite, yet small patch of moist desquamation was seen centrally. Cobalt therapy was completed September 22. By October 22, 1953, there was a fetid odour to the breath with oedema and suspicion of cartilage necrosis. Some four and one-half months later, during the week of March 14, 1954, a tracheotomy was performed and several biopsies obtained. Unfortunately, one was taken through the skin over the left thyroid cartilage and subsequently a fistula developed in this region. The biopsies were all negative. The tracheotomy spontaneously sealed, had to be re-opened and when seen in February 1956 there was still a laryngeal fistula with marked telangiectasia and some oedema and granularity of the larynx. There was no evidence of disease when last seen but the fistula persisted.

In commenting on this case of cartilage necrosis one can say that the dose was moderate. The fields were average in size. Reaction was maximum both on one small patch of skin and throughout the mucosa and we associate necrosis in this case primarily with previous heavy x-radiation. Biopsies through the skin probably contributed to fistula.

Case D. S. #52 882, age 55. The right cord was replaced by tumour and fixed. The epiglottis was encroached upon posteriorly and deformed. The stage was 2. The histological grading was II. Cobalt^{60} beam therapy was commenced July 8, 1952. Tomography in July 1952 showed very gross swelling of the right wall of the larynx: this abnormality seemed to extend downward beneath the glottis for 7 cm. There was a suggestion of partial destruction of the thyroid cartilage on the right side. Probably because of the radiographic suggestion of downward extension, huge fields were taken: 10x17 cm.

posterolaterally and an anterior field of similar length but somewhat narrower. 5000 roentgens were delivered to the larynx over a period of twenty-nine days. The patient was not seen for five months but when examined in January 1953 his mouth was dry, the voice was still husky and oedema beneath mandible extended subcutaneously into either side of neck. There was moderate oedema of both arytenoids and false cords. The true cords were mobile. There was no evidence of active malignancy. In March 1953, the patient had a coronary thrombosis. When seen in May 1953 he had tenderness over the left thyroid cartilage; necrosis was suspected but the attending otolaryngologist hesitated to biopsy. By September 1953 the patient required tracheotomy. He died January 1954 with the recorded death of cancer of the larynx.

In commenting on this second case of cartilage necrosis one might say that the volume treated was 8 cm. in diameter and 17 cm. in length. We are accepting this case as too high a dose for the volume treated but we are cognizant of the fact that the X-ray showed suspicious cartilage invasion when first seen. There was no final proof of persistent cancer.

Illustration of Repeated Treatment

Case J. P. #52/640, age 57. This patient, who was a diabetic, presented with a carcinoma of the right true and false cords, with fixation which was not complete. A three-field technique was used, the portals being 5x6 cm. anteriorly, 6x6 cm. posterolaterally, and a tumour dose of 4500 r in an overall period of three weeks was given. There was a heavy fibrinous reaction upon completion, with considerable dysphagia and although a tumour dose of 4500 r is considered a lethal borderline, the patient was none too co-operative and clinically we felt it might be risky to go on. Treatment was completed on June 6, 1952. Five months later, November 18, 1952, a recurrence was proven histologically. The patient refused laryngectomy and we accepted for a single field 8x6 cm. delivering a tumour dose of 5000 roentgens in twenty days. Three and one-half years later the patient reports a voice which is almost normal. His doctor reports no active disease.

This case presents a surprising restraint, or we hope, cure, from a second Cobalt series following initial failure. This is unique in our experience but laryngectomy was flatly refused, the patient being an attorney said he had to earn his living by his voice. We accepted the challenging, yet realistic situation by treating a second time.

Late Effects

From follow-up observations of the surviving group, one can say that there is a tendency to dryness of laryngeal mucosa, huskiness of voice and, as a rule, some minimal residual laryngeal oedema. The odd case shows telangiectasia of the mucous membrane. These changes have presented no unusual difficulties to date. In no instance was tracheotomy required during treatment. Two cases required tracheotomy later, both of which are reported in detail previously.

The treated skin remains pliable and soft, the occasional case showing minimal anterior telangiectasia. There is nothing to suggest that subsequent laryngectomy, if necessary, would be impaired by previous Cobalt^{60} irradiation.

Summary

A small, yet we believe, significant series of intrinsic carcinoma of the larynx treated by Cobalt^{60} beam therapy is presented with reference particularly to dosage studies and clinical response. The time period is too short and the case volume too small to deduce from a statistical viewpoint. Eleven cases out of thirteen are without disease thirteen months to four and one-half years; one case, well when last seen a year after treatment is now untraced; one died from his disease.

Standard Cobalt^{60} technique was by plaster of paris jacket, three-field beam direction, one anterior and two posterolateral fields. The average field sizes were 5x5 cm., 6x5 cm., the largest being up to 10x17 cm. Tumour doses ranged from 4500 r to 7000 r in three to five weeks.

It is our impression that with the dose rate used, fibrinous reactions appeared earlier and regressed more rapidly than when treated by X-ray. It is to be remembered however, that the dose rate was higher and the overall time shorter than with our X-ray therapy group. Only occasionally did a patch of moist dermatitis occur anteriorly. Dysphagia, usually to a mild degree, was seen, occasionally severe but short-lived.

Cartilage necrosis appeared in one case which years previously had received X-ray therapy, and in a second case when extremely large fields, 10x17 cm., were used. The first case mentioned is well with laryngeal fistula: the second case died, probably with extension of disease.

Late observations show that treated skin remains pliable and soft, only occasionally showing minimal anterior telangiectasia. There is a tendency to dryness of laryngeal mucosa, huskiness of voice and often some minimal residual laryngeal oedema.

It is apparent in the intrinsic group, that surgery should be held in abeyance for complications or recurrence following Cobalt^{60} beam therapy. Six of this group showing subglottic extension radiographically, all with disease controlled except one, lead us to believe, contrary to accepted teaching, that laryngectomy is not the prime method of attack in the larynx lesion with subglottic extension.

DOSE MEASUREMENTS IN THE BUILD-UP REGION FOR COBALT^{60} RADIATION

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Toronto, Ontario

Introduction

Depth dose measurements in the build-up region of an absorbing medium exposed to high energy radiation have been made by a number of workers. Johns has reported depth dose values for Cobalt^{60} [6] radiation and for 22 MeV radiation[5] while Howarth[4] and Trump[10] have measured build-up curves for 2 MeV and 3 MeV X-rays respectively. All of these investigators have shown that the absorbed dose rises from a low value at the surface of the medium to a maximum value at a depth below the surface. The depth of this maximum increases with the energy of the beam. For Cobalt^{60} radiation the maximum dose occurs between depths of 4 and 5 mm. below the surface. The value of the dose on the surface relative to the maximum dose has been shown to be very dependent on the physical geometry[6].

The radiation absorbed in the build-up region is usually measured by a thin window, parallel plate ionization chamber mounted in the surface of a phantom. Thin sheets of absorber placed over the thin window serve to increase the depth of the chamber in the phantom, so that the dose at each depth can be measured. It has often been observed that dosage measurements made in this manner are dependent upon the polarity of the collecting voltage across the ionization chamber. This polarity effect, as it is called, is greatest near the surface and decreases with depth and vanishes at the depth of the maximum dose. Richardson[8] has shown that the polarity effect is caused by the interaction of the high energy radiation with the ion chamber itself. In this paper, a description of the polarity effect is given. A thin window ionization chamber is described which does not exhibit the polarity effect, and some measurements made with this chamber on an A.E.C.L. Model A Cobalt unit and a Picker ceiling mount unit are presented.

Experiments

Typical build-up curves illustrating the polarity effect are shown in Figure 1. The upper curve was obtained when positive ions were collected and yields a surface dose of 27% of the maximum dose. The current, obtained when negative charges are collected, is shown by the lower curve and gives a surface dose of 12%. The surface ionization ratio (defined as the ratio of the surface dose to the maximum dose) for these two cases differs by a factor of more than 2. The question arises as to which is the true surface dose. A number of people have taken the average as the true surface ionization and this will be shown to be the correct procedure.

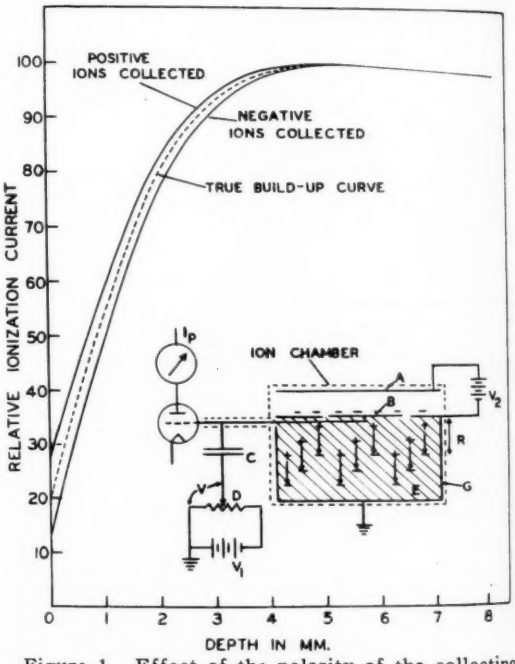

Figure 1. Effect of the polarity of the collecting voltage on "build-up" curves for Cobalt^{60} radiation.

A schematic diagram of the type of ionization chamber used to obtain these results is shown as the insert of Figure 1. The upper electrode A is made of a thin sheet of polythene film coated on the inside with aquadag. The lower electrode E is made of polystyrene and is coated over its complete outer surface by a thin layer of aquadag. A circular groove is inscribed through the aquadag on the upper surface to separate the collecting electrode B from the guard ring surrounding it. The central electrode B is connected to the grid of an electrometer tube through a shielded cable while the conducting layer on the outside of E is held at ground potential. The

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grounded external layer on the electrode serves to shield the electrode B. The grid of the electrometer is connected to the compensating condenser C. The lower terminal of the condenser C is connected to the potential divider. The electrode B is held at ground potential as the exposure proceeds by sliding the contact D from left to right. The electrode is held at ground potential by keeping the plate current, I_p, in the electrometer tube, constant. Thus, during the exposure, electrode B is always at ground potential. In the diagram the collecting voltage of electrode A is shown as positive and is represented by V_2. When positive ions are being collected, the polarity of the compensating voltage V_1 must be as indicated. If V_2 is reversed so that negative charges are collected, then V_1 must also be reversed. The charge collected in both cases is CV.

The interaction of the radiation with the electrode E is shown schematically. When high energy radiation interacts with matter, electrons are ejected primarily in the forward direction and in this diagram are represented as going forward a distance R. Thus, for each interaction, we obtain a positive charge at the site of interaction and a negative charge deposited where the electron comes to rest. In a dielectric, these charges will remain in these positions for a long period of time. From the diagram, it is obvious that at a depth equal to the range R below the surface, there will be no net charge produced in the dielectric but between this plane and the surface, there will be a continual increase in positive charge density to a maximum value at the surface. These positive charges will induce negative charges on the electrode B which will remove negative charges from the upper plate of C leaving a net positive charge on C. The charge deposited on the upper electrode C will thus be $Q_+ = Q_i + Q_d$ where Q_i is the charge produced by ionization in the air cavity and Q_d is the charge induced in the dielectric by the charge distribution described above. If we reverse the polarities of V_2 and V_1 and repeat the experiment, it is readily seen that the charge collected will now be $Q_- = Q_i - Q_d$. It is conceivable that in a chamber containing a thick dielectric and a very thin air volume, Q_- could be negative. In a detailed discussion, to be described elsewhere[9], theoretical expressions for Q_d have been obtained in terms of the dimensions of the ion chamber. Consider now what happens when the front electrode A is made thicker. If it is made equal to the average range of the electrons R, then there is no net production of charge in the electrode E and the polarity effect disappears as indicated in Figure 1.

In the case described above only a fraction of the positive charges produced in the dielectric induce negative charges on the electrode B. If, however, the electrode E is made of a conducting material, then the total charge produced in the electrode is collected. The size of the dielectric current, Q_d, depends upon the area of the electrode B, the thickness of the electrode and also upon the energy of the radiation. Details of experiments to investigate these effects are given elsewhere[9].

From Figure 1 it is obvious that the dielectric current can be reduced to a small value if the electrode E is made very thin. The construction of such a chamber, however, presents many practical difficulties. A chamber which we have found satisfactory is shown in Figure 2. It consists of three thin electrodes stretched across four interlocking polystyrene rings. The upper and lower electrodes are made with polythene coated with aquadag. The central electrode was constructed by stretching a nylon stocking over the centre

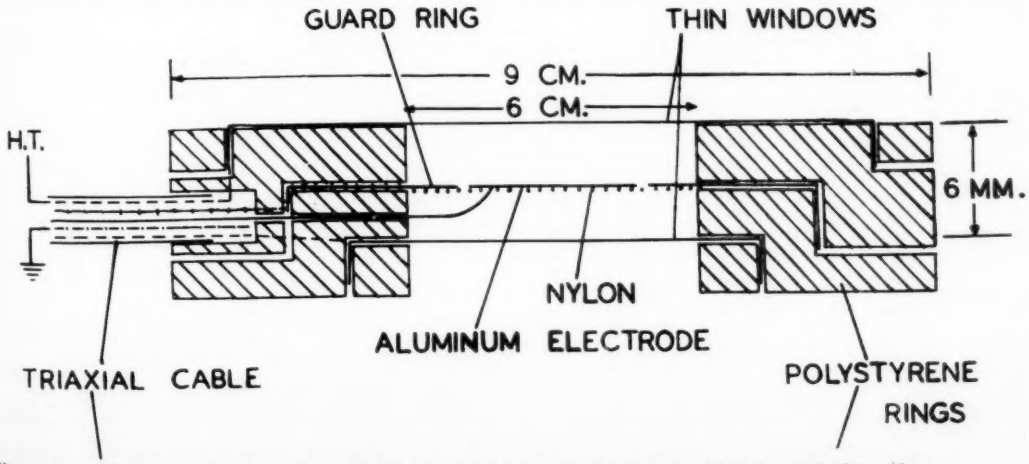

Figure 2. Schematic diagram of an ionization chamber which does not show any polarity effect.

ring of polystyrene and sticking to this nylon net a circular collecting electrode made of very thin aluminum surrounded by an annular ring of the same material which forms the guard ring. Connections to the central electrode were made by means of a fine wire extending through the lower volume of the chamber and fastened to the aluminum electrode. Details of the chamber are shown in the diagram. Such a chamber was found to show no polarity effect. Since the collecting electrode is now very thin, the thickness of the lower electrode is of no great significance and it could have been made very thick. However, the chamber was designed symmetrically so that the device could be turned over in the beam. To measure a build-up curve with such a chamber, it was mounted on a suitable phantom and then layers of material were added to the upper surface. Typical results are given in Table I. It is apparent that ion current is independent of polarity.

Measurements were made on a Picker ceiling mounted Cobalt unit with a multi-vane collimator[7] and with the A.E.C.L. Model A unit[3a, b]. The surface dose was found to be a function of the particular geometry of the unit. In general the surface dose is smallest with the small fields and increases with increase in field size.

Results

The A.E.C.L. machine is supplied with three possible arrangements for localizing the field.

TABLE I

TO ILLUSTRATE THE ABSENCE OF POLARITY EFFECT WITH THE CHAMBER ILLUSTRATED IN FIG. 2

Thickness of Absorber over ion chamber mg/cm²	Dose as a Percentage of the Maximum*	
	Collection positive ions	Collection negative ions
0	15.5	15.5
17	27.0	27.1
34	35.5	35.5
51	43.5	43.4
85	57.0	56.8
170	80.3	80.5
255	93.3	94.0
340	99.5	99.0
391	99.8	100.2
425	99.5	99.7
595	99.5	99.2
850	98.7	98.7
1700	94.5	94.5

*The maximum is taken as the average of the readings obtained with +ve and —ve voltage for a front wall of 391 mg/cm².

(a) Open-ended lucite cones which may be attached to the machine. These cones are slightly larger than the field size so that the direct beam does not strike the cone.

(b) The machine may be used with no attachments if a distance localizer similar to the one used on the Picker unit is employed.

(c) A universal pointed lucite cone which marks the treatment distance and the center of the field and may be used with any field size. (See Figure 4 of reference 3a.)

The Picker ceiling mount unit using the multivane collimator[7] may be used in ways (a) and (b) described above. The collimator is provided with a 1 mm thick, clear, lucite dust cover which slips into the end of the collimator.

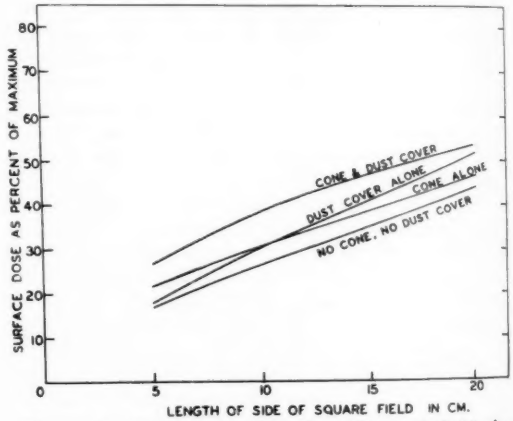
SURFACE DOSE AS PERCENT OF MAXIMUM
LENGTH OF SIDE OF SQUARE FIELD IN CM.
CONE & DUST COVER
DUST COVER ALONE
CONE ALONE
NO CONE, NO DUST COVER

Figure 3. Graphs showing the effect of field size on the surface dose for a Picker Cobalt⁶⁰ unit. Measurements were made with and without "dust cover" and treatment cones as indicated.

Some results for the Picker unit are shown in Figure 3 for four cases, (a) and (b), with and without the dust cover. In Figure 3 the surface dose is plotted as a function of the length of the side of the square field. The lowest surface dose is obtained when no cone and no dust cover are used. In all cases the surface dose increases with field size. The dust cover is unnecessary and may be dispensed with, or replaced by a much thinner layer of material.

Transit dose measurements were made on the Picker unit by moving the ion chamber along one axis of a 20 x 20 cm field. The dose on the surface (expressed as a percentage of the maximum dose) is plotted against the distance from the centre of the field in Figure 4. The lower curve, obtained using no cone,

shows that initially the surface dose decreases slowly with distance from the centre of the field. The decrease in dose becomes more rapid near the edge of the field. The upper curve of Figure 4 was measured using an open-ended cone. The surface dose at every point is greater than the dose obtained using no cone. These measurements indicate that the maximum surface contamination is at the centre of the field.

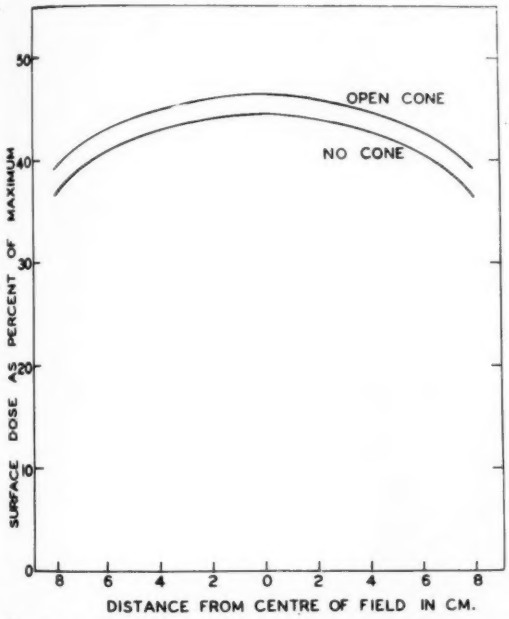

Figure 4. Variation of dose across the surface of a 20 x 20 cm field for a Picker Cobalt^{60} unit.

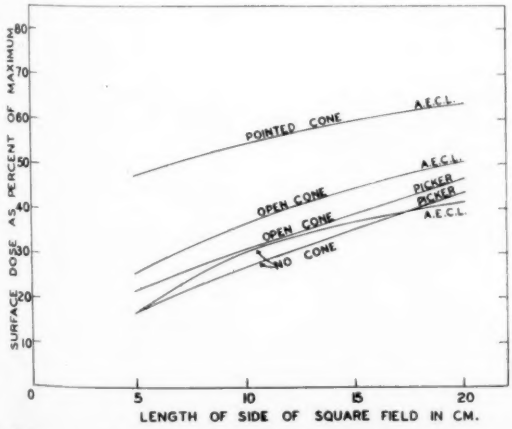

Figure 5. Comparison of surface doses for Picker and A.E.C.L. Cobalt^{60} units, showing the effects of field size and the type of treatment cones.

The Picker unit, without the dust cover, is compared with the A.E.C.L. unit in Figure 5. When no cone is used the two units give essentially the same surface dose. When an open-ended cone is used, the Picker unit gives a slightly lower surface dose. The A.E.C.L. unit using the pointed cone gives a very much higher surface dose. Two papers indicating the clinical importance of maintaining a low surface dose have been presented in the literature by Burkell[2] and Batley[1]. We conclude that where possible the units should be used without cones, and the pointed cone of the A.E.C.L. unit should never be used. The physical advantages of high energy radiation should not be lightly discarded through the use of incorrectly designed treatment cones.

Summary

(a) In order to minimize surface dose, the pointed cone supplied with the A.E.C.L. Model A unit should not be used.

(b) The dust cover on the Picker unit should be removed.

(c) Where possible, both units should be used without cones.

Acknowledgments

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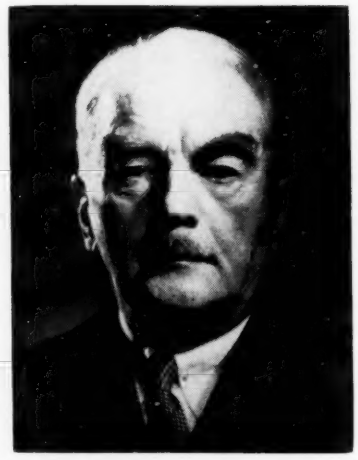
THE REWARD OF A THING WELL DONE IS TO HAVE DONE IT — Emerson

Reflections from the 20th year plateau of our Association beam signal honours on the late Gordon E. Richards for his inspirational imagination as founder. It was in 1937, on January 4th and 5th, that, heeding his invitation to all Canadian Radiologists, the first session came into assembly at the Toronto General Hospital.

In response to present interest to know more of the organizing and continuing contributors, the first slate of officers is here recalled; and the founder, the first and all succeeding presidents are given pictorial illumination; yet this is not to forget the superlative secretaries of the years as: E. M. Crawford, D. L. McRae, Guillaume Gill, and the late A. D. Irvine and W. L. Ritchie.

At the first meeting Dr. Richards declined the Presidency, and it was the unanimous opinion that his founder's role transcended this office.

The 1937 slate of officers was: President, W. A. Jones, Kingston; Vice President, J. E. Gendreau, Montreal; Secretary, A. C. Singleton, Toronto. The Councillors were: C. W. Prowd, Vancouver; W. H. McGuffin, Calgary; C. M. Henry, Regina; Digby Wheeler, Winnipeg; W. M. Gilmore, Stratford; Paul Brodeur, Ottawa; E. H. Shannon, Toronto; Lloyd Ritchie, Montreal (English); L. A. Gagnier, Montreal (French); J. E. Perron, Quebec City; S. R. Johnston, Halifax; A. S. Kirkland, Saint John, N.B.; and Gordon E. Richards was the Chairman of the Committee on Inter-relations.



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1939 - 1940

W. H. McGUFFIN*
1941 - 1943

J. E. PERRON*
1944

*Deceased

M. C. MORRISON*
1945

C. W. PROWD*
1946

DIGBY WHEELER*
1947

A. C. SINGLETON
1948

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1949

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1950

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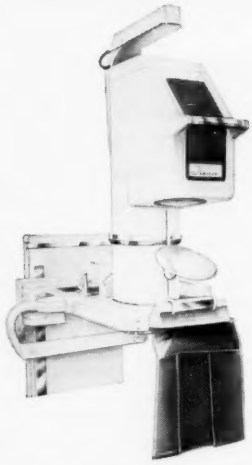

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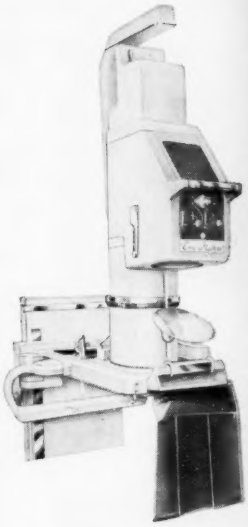

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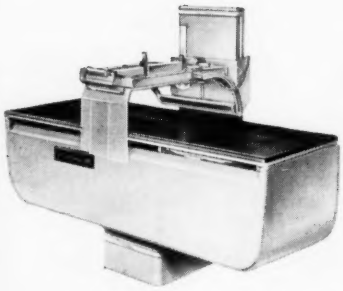

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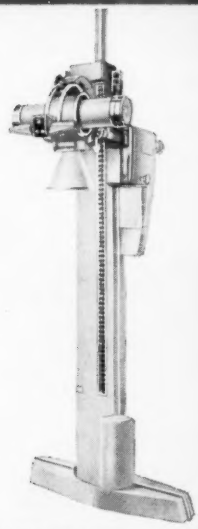

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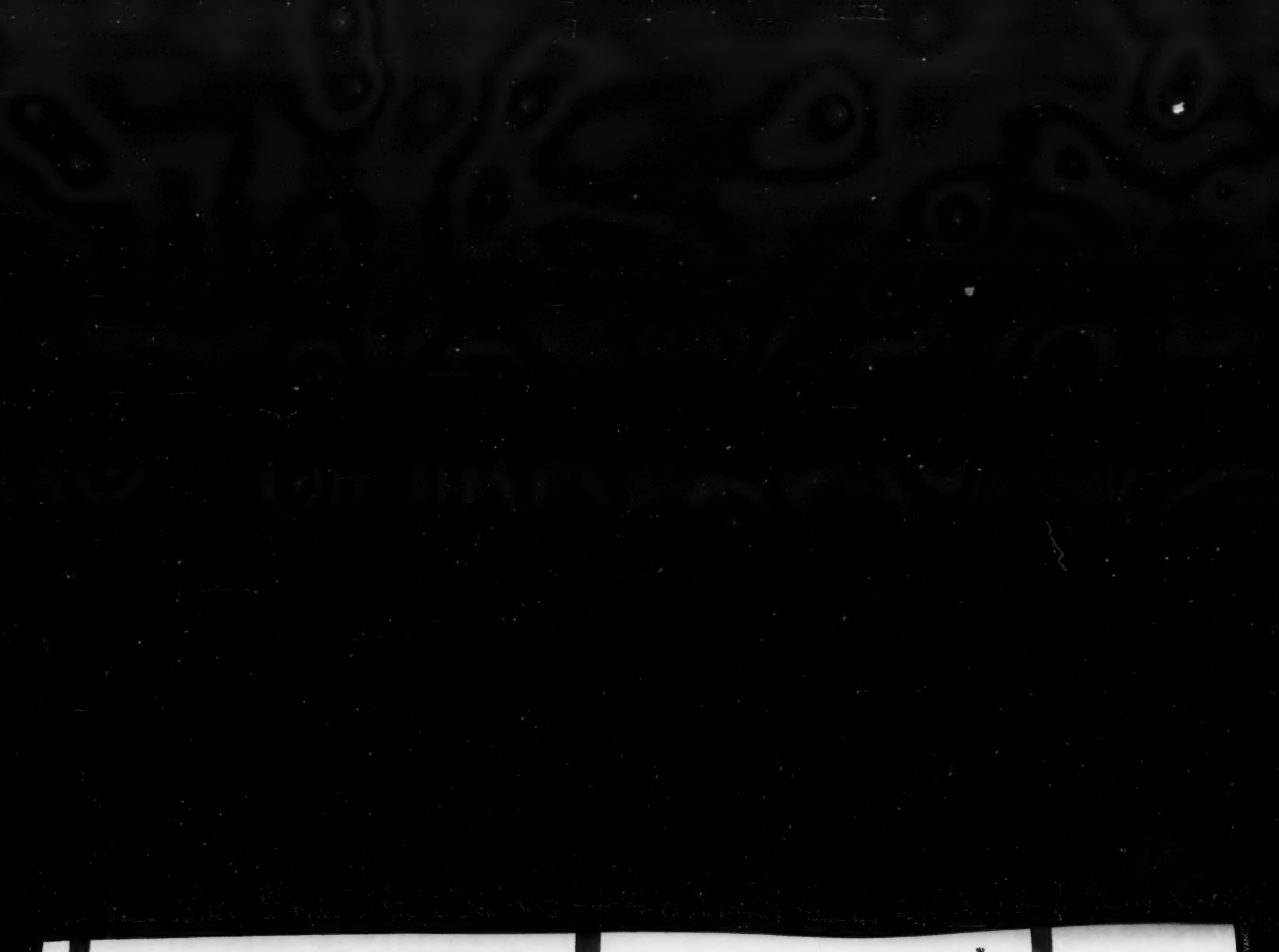
261 DAVENPORT ROAD, TORONTO, ONTARIO

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